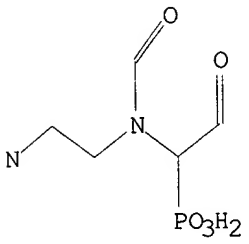
=> d que stat l1
L1 STR



Structure attributes must be viewed using STN Express query preparation.

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(FILE 'HOME' ENTERED AT 02:57:08 ON 07 FEB 2003)

FILE 'REGISTRY' ENTERED AT 02:57:16 ON 07 FEB 2003
L1 STRUCTURE UPLOADED
L2 0 L1 SSS FULL

FILE 'AGRICOLA, ALUMINIUM, ANABSTR, APOLLIT, AQUIRE, BABS, BIOCOMMERCE, BIOTECHNO, CABA, CAOLD, CAPLUS, CBNB, CEABA-VTB, CEN, CERAB, CIN, COMPENDEX, CONFSCI, COPPERLIT, CORROSION, ENCOMPLIT, ENCOMPLIT2, FEDRIP, GENBANK, INSPEC, INSPHYS, INVESTEXT, IPA, ...' ENTERED AT 03:03:49 ON 07 FEB 2003

L4 771 ((PEPTIDE(W)NUCLEIC(W)ACID) OR PNA OR (AMINOETHYL(W)GLYCINE)) A
L5 125 L4 NOT PY>=1999
L6 125 DUP REM L5 (0 DUPLICATES REMOVED)
L7 102 ((PEPTIDE(W)NUCLEIC(W)ACID) OR PNA OR (AMINOETHYL(W)GLYCINE)) (
L8 98 DUP REM L7 (4 DUPLICATES REMOVED)
L9 11 L8 NOT PY>=1999

=> d l9 total ibib abs

L9 ANSWER 1 OF 11 LIFESCI COPYRIGHT 2003 CSA
ACCESSION NUMBER: 94:35142 LIFESCI
TITLE: Peptide nucleic acids (PNA). Oligonucleotide analogues with an achiral peptide backbone
AUTHOR: Egholm, M.; Buchardt, O.; Nielsen, P.E.; Berg, R.H.
CORPORATE SOURCE: Res. Cent. Med. Biotechnol., Chem. Lab. II, H.C. Oersted Inst., Univ. Copenhagen, Universitetsparken 5, DK-2100 Copenhagen Oe, Denmark
SOURCE: J. AM. CHEM. SOC., (1992) vol. 114, no. 5, pp. 1895-1897.
DOCUMENT TYPE: Journal
FILE SEGMENT: N
LANGUAGE: English
AB Oligonucleotides that specifically recognize messenger RNA or double-stranded DNA present unique opportunities for inhibiting protein synthesis (the antisense approach) or for modulating gene expression, e.g., via triple helix formation. The deoxyribose phosphate backbone of DNA has been modified in a number of ways to increase nuclease stability and cell membrane permeability, assuming that major changes would be deleterious to the "DNA hybridization properties". Consequently, derivatives such as mono- or dithiophosphates, methyl phosphonates, **borano** phosphates, etc., as well as formacetal, carbamate, and siloxane, or dimethylenethio-, -sulfoxido-, and -sulfono-linked, species were prepared. The synthesis of peptides is more versatile than oligonucleotide synthesis, allowing the facile design of an achiral backbone and relatively large-scale production. We therefore designed **peptide nucleic acids** (**PNA**), i.e., molecules where the individual nucleobases were linked to an achiral peptide backbone.

L9 ANSWER 2 OF 11 USPATFULL
ACCESSION NUMBER: 1998:134849 USPATFULL
TITLE: P-glycoprotein mutant resistant to cyclosporin modulation
INVENTOR(S): Sikic, Branimir I., Stanford, CA, United States
Chen, Gang, Palo Alto, CA, United States
PATENT ASSIGNEE(S): The Board of Trustees of the Leland Stanford Junior University, Palo Alto, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5830697		19981103	
APPLICATION INFO.:	US 1997-784649		19970121	(8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted

PRIMARY EXAMINER: Carlson, Karen C.
LEGAL REPRESENTATIVE: Sherwood, Pamela J.Bozicevic & Reed LLP
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
LINE COUNT: 1174
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A mutated form of human P-glycoprotein (mdr1δF335/336) is identified, consisting of a single or double codon deletion (Phe335 and/or 336) in the TM region of P-gp. The mdr1δF335/336 encoded P-glycoprotein is characterized by an altered spectrum of cross-reactivity to cytotoxins and resistance to modulation by cyclosporins, with a loss of the capacity to bind or transport cyclosporine, PSC 833, and vinblastine. These data demonstrate that cyclosporine, PSC 833, vinblastine, Rh-123, and dactinomycin share at least one binding domain on, which plays an important role in the interaction of P-gp with modulators. The nucleic acid compositions encoding mdr1δF335/336 find use in gene therapy to transfer modulator-resistant multidrug resistance into transfected cells; to produce the encoded protein for functional mapping studies, and in studying associated physiological pathways.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 3 OF 11 USPATFULL
ACCESSION NUMBER: 1998:134811 USPATFULL
TITLE: Oligonucleotide sizing using cleavable primers
INVENTOR(S): Monforte, Joseph Albert, Berkeley, CA, United States
Becker, Christopher Hank, Menlo Park, CA, United States
Shaler, Thomas Andrew, San Francisco, CA, United States
Pollart, Daniel Joseph, Menlo Park, CA, United States
PATENT ASSIGNEE(S): SRI International, Menlo Park, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5830655		19981103
APPLICATION INFO.:	US 1996-639363		19960426 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1995-445751, filed on 22 May 1995
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Horlick, Kenneth R.
ASSISTANT EXAMINER: Tung, Joyce
LEGAL REPRESENTATIVE: Evans, Susan T., Fabian, Gary R.
NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 59 Drawing Figure(s); 24 Drawing Page(s)
LINE COUNT: 3411
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides modified oligonucleotide primers designed to incorporate a cleavable moiety so that a 3' portion of the primer (linked to an extension product) can be released from an upstream 5' portion of the primer. Upon selective cleavage of the cleavable site, primer extension products that contain about five or fewer base pairs of the primer sequence are released, to provide more useful sizing and sequence information per fragment than extension products containing the entire primer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 4 OF 11 USPATFULL

ACCESSION NUMBER:	1998:79147 USPATFULL
TITLE:	Apoptotic regression of intimal vascular lesions
INVENTOR(S):	Gibbons, Gary H., Palo Alto, CA, United States Pollman, Matthew J., San Francisco, CA, United States
PATENT ASSIGNEE(S):	The Board of Trustees of the Leland Stamford Junior University, Palo Alto, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5776905		19980707	
APPLICATION INFO.:	US 1996-694927		19960808	(8)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	Granted
PRIMARY EXAMINER:	Marschel, Ardin H.
LEGAL REPRESENTATIVE:	Sherwood, Pamela J.Bozicevic & Reed LLP
NUMBER OF CLAIMS:	11
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	2 Drawing Figure(s); 1 Drawing Page(s)
LINE COUNT:	769

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Neointimal cells are shown to express high levels of an anti-apoptotic gene, bcl-x, while the medial cells of the vessel itself express only low levels. The difference in gene expression exploited to provide for selective deletion of the neointimal cells. Apoptosis is induced by administering anti-sense bcl-x oligonucleotides to the affected vessel. Apoptosis is desirable as a treatment because it does not induce inflammation, further tissue injury or reactive hyperplasia. A significant reduction in lesion size is seen after treatment.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 5 OF 11 USPATFULL

ACCESSION NUMBER:	1998:79009 USPATFULL
TITLE:	Obesity associated genes
INVENTOR(S):	North, Michael, La Jolla, CA, United States Nishina, Patsy, Bar Harbor, ME, United States Noben-Trauth, Konrad, Bar Harbor, ME, United States Naggert, Juergen, Bar Harbor, ME, United States
PATENT ASSIGNEE(S):	Sequana Therapeutics, Inc., La Jolla, CA, United States (U.S. corporation) The Jackson Laboratory, Bar Harbor, ME, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5776762		19980707	
APPLICATION INFO.:	US 1996-714991		19960917	(8)

RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1996-630592, filed on 10 Apr 1996
DOCUMENT TYPE:	Utility
FILE SEGMENT:	Granted
PRIMARY EXAMINER:	Chambers, Jasemine C.
ASSISTANT EXAMINER:	Priebe, Scott D.
LEGAL REPRESENTATIVE:	Sherwood, Pamela J.Bozicevic & Reed LLP
NUMBER OF CLAIMS:	4
EXEMPLARY CLAIM:	1,4

NUMBER OF DRAWINGS: 1 Drawing Figure(s); 1 Drawing Page(s)
LINE COUNT: 1483
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The gene responsible for the autosomal recessive mouse obesity mutation tub was identified by positional cloning. The homologous human gene is also provided. The genes are used to produce tubby protein; in screening for compositions that modulate the expression or function of the tubby protein; and in studying associated physiological pathways. The DNA is further used as a diagnostic for genetic predisposition to obesity, retinal degeneration or cochlear degeneration. The mutation responsible for the tub phenotype is a G to T transversion that abolishes a donor splice site in the 3' coding region and results in a larger transcript containing the unspliced intron. A second, prematurely truncated transcript arises from the introduction of a premature polyadenylation site in the unspliced intron.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 6 OF 11 USPATFULL
ACCESSION NUMBER: 1998:61431 USPATFULL
TITLE: Mutant human hedgehog gene
INVENTOR(S): Epstein, Ervin, Orinda, CA, United States
Hu, Zhilan, San Francisco, CA, United States
Bonifas, Jeanette, San Francisco, CA, United States
PATENT ASSIGNEE(S): The Regents of the University of California, Oakland, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5759811		19980602	
APPLICATION INFO.:	US 1996-748591		19961113	(8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Wax, Robert A.
ASSISTANT EXAMINER: Stole, Einar
LEGAL REPRESENTATIVE: Sherwood, Pamela J.Bozicevic & Reed LLP
NUMBER OF CLAIMS: 17
EXEMPLARY CLAIM: 1
LINE COUNT: 1271
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A mutation in the human sonic hedgehog gene is associated with tumorigenesis. A variety of human tumors, including basal cell carcinomas, breast carcinomas, medulloblastomas, etc., have a somatic mutation that results in an amino acid substitution at position 133 [*his133 SHH], or in a mutation at position 114. Such mutated genes and fragments thereof, encoded protein, and antibodies specific for the mutated protein are useful in characterizing the phenotype of associated tumors. The mutant protein is useful in drug screening for compositions that antagonize or otherwise modulate HH activity or expression. The encoded protein is also used as a therapeutic, to modulate cell proliferation and differentiation, and treatment of pathological conditions associated with decreased hedgehog signaling.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 7 OF 11 USPATFULL
ACCESSION NUMBER: 1998:45195 USPATFULL
TITLE: Combination for treatment of proliferative diseases
INVENTOR(S): Muller, Marcel, Allschwil, Switzerland

Geiger, Thomas, Freiburg, Germany, Federal Republic of
Altmann, Karl-Heinz, Reinach, Switzerland
Fabbro, Doriano, Arlesheim, Switzerland
Dean, Nicholas M., Encinitas, CA, United States
Monia, Brett, Carlsbad, CA, United States
Bennett, Clarence Frank, Carlsbad, CA, United States

PATENT ASSIGNEE(S): Novartis Corporation, Summit, NJ, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5744460		19980428	
APPLICATION INFO.:	US 1996-612775		19960307	(8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Robinson, Douglas W.
ASSISTANT EXAMINER: Nelson, Amy J.
LEGAL REPRESENTATIVE: Nowak, Henry P.
NUMBER OF CLAIMS: 12
EXEMPLARY CLAIM: 1
LINE COUNT: 2910
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention relates to combinations of PKC-targeted (especially PKC-α-targeted) deoxyribo- and ribo-oligonucleotides and derivatives thereof with other chemotherapeutic compounds, as well as to pharmaceutical preparations and/or therapies, in relation to disease states which respond to such oligonucleotides or oligonucleotide derivatives, especially to to modulation of the activity of a regulatory protein. In particular, the invention relates to products or combinations comprising antisense oligonucleotides or oligonucleotide derivatives targeted to nucleic acids encoding human PKC and other (preferably standard) chemotherapeutics, either in fixed combination or for chronologically staggered or simultaneous administration, and the combined use of both classes of compounds, either in fixed combination or for chronologically staggered or simultaneous administration, for the treatment of proliferative diseases, especially tumor diseases, that can be treated by inhibition of PKC activity, that is, where the antisense oligonucleotides or oligonucleotide derivatives are targeted to nucleic acids encoding the regulatory protein PKC or active mutated derivatives thereof.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 8 OF 11 USPATFULL
ACCESSION NUMBER: 1998:1667 USPATFULL
TITLE: Identification of a gene encoding TULP2, a retina specific protein
INVENTOR(S): North, Michael, San Diego, CA, United States
Nishina, Patsy, Bar Harbor, ME, United States
Naggert, Juergen, Bar Harbor, ME, United States

PATENT ASSIGNEE(S): Sequana Theraputics, Inc., La Jolla, CA, United States (U.S. corporation)
Jackson Lab., Bar Harbor, ME, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5705380		19980106	
APPLICATION INFO.:	US 1996-706292		19960904	(8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Jones, W. Gary
ASSISTANT EXAMINER: Shoemaker, Debra
LEGAL REPRESENTATIVE: Sherwood, Ph. D., PamelaBozicevic & Reed, LLP.
NUMBER OF CLAIMS: 6
EXEMPLARY CLAIM: 1,3,4
LINE COUNT: 942
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The gene responsible for an autosomal dominant con-rod retinal dystrophy is identified, TULP2. The genes are used to produce the encoded protein; in screening for compositions that modulate the expression or function of TULP2 protein; and in studying associated physiological pathways.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 9 OF 11 USPATFULL
ACCESSION NUMBER: 97:109710 USPATFULL
TITLE: Methods for combined PCR amplification and hybridization probing using doubly labeled fluorescent probes
INVENTOR(S): Mayrand, Paul E., Pacifica, CA, United States
PATENT ASSIGNEE(S): The Perkin Elmer Corporation, Foster City, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5691146		19971125
APPLICATION INFO.:	US 1996-710075		19960911 (8)

RELATED APPLN. INFO.: Division of Ser. No. US 1995-435509, filed on 5 May 1995
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Jones, W. Gary
ASSISTANT EXAMINER: Fredman, Jeffrey
LEGAL REPRESENTATIVE: Grossman, Paul D.
NUMBER OF CLAIMS: 10
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Figure(s); 2 Drawing Page(s)
LINE COUNT: 748
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB An oligonucleotide probe is disclosed, the probe including an oligonucleotide, a fluorescer molecule attached to a first end of the oligonucleotide and a quencher molecule attached to the opposite end of the oligonucleotide. The probe is rendered impervious to digestion by the 5'→3' exonuclease activity of a polymerase and the 3'→5' extension of by a polymerase. The invention also includes methods for performing combined PCR amplification and hybridization probing, one such method including the steps of contacting a target nucleic acid sequence with PCR reagents and an oligonucleotide probe as described above, and subjecting these reagents to thermal cycling. One preferred refinement of the above method further includes the addition of a strand displacer to facilitate amplification. Additional similar combined PCR hybridization methods are disclosed, such methods not requiring probes having their 5' ends protected, wherein (i) the polymerase lacks 5'→3' exonuclease activity, (ii) a 5'→3' exonuclease inhibitor is included, and (iii) an exonuclease reactivation step is performed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 10 OF 11 USPATFULL

ACCESSION NUMBER: 97:104620 USPATFULL
TITLE: Genes associated with retinal dystrophies
INVENTOR(S): North, Michael, San Diego, CA, United States
Nishina, Patsy, Bar Harbor, ME, United States
Naggert, Juergen, Bar Harbor, ME, United States
PATENT ASSIGNEE(S): The Jackson Laboratory, Bar Harbor, ME, United States (U.S. corporation)
Sequana Therapeutics, Inc., La Jolla, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5686598		19971111	
APPLICATION INFO.:	US 1996-701380		19960822	(8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Chan, Christina Y.
ASSISTANT EXAMINER: VanderVegt, F. Pierre
LEGAL REPRESENTATIVE: Fish & Richardson P.C., Sherwood, Pamela J.
NUMBER OF CLAIMS: 2
EXEMPLARY CLAIM: 1
LINE COUNT: 889
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The gene responsible for the autosomal recessive retinal degenerative disease RP 14 is identified, TULP1. The genes are used to produce the encoded protein; in screening for compositions that modulate the expression or function of TULP1 protein; and in studying associated physiological pathways. The DNA is further used as a diagnostic for genetic predisposition to retinal degeneration.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 11 OF 11 USPATFULL

ACCESSION NUMBER: 97:42420 USPATFULL
TITLE: Boron neutron capture enhancement of fast neutron therapy
INVENTOR(S): Griffin, Brian R., Edmonds, WA, United States
Laramore, George E., Seattle, WA, United States
PATENT ASSIGNEE(S): IONIX Corporation, Seattle, WA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5630786		19970520	
APPLICATION INFO.:	US 1994-267350		19940627	(8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: McDermott, Corrine M.
ASSISTANT EXAMINER: Smith, Chalin
LEGAL REPRESENTATIVE: Christensen O'Connor Johnson & Kindness PLLC
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Figure(s); 1 Drawing Page(s)
LINE COUNT: 953

AB Fast neutron therapy is significantly enhanced by irradiating target cells, either in vitro or in vivo, with fast neutrons in the presence of

a boron neutron capture agent having a plurality, and preferably at least nine, of .sup.10 B atoms per molecule of the agent. In other aspects, tumor cells are treated in vivo by administering to a human or non-human animal a boron neutron capture agent having at least nine .sup.10 B atoms per molecule of the agent in an amount effective to associate the boron neutron capture agent with the tumor cells, and then irradiating the tumor cells with fast neutrons. Suitable boron neutron capture agents may be based on polyhedral borane anion derivatives, on derivatives that comprise two polyhedral borane anion cages linked together to form a structure comprising 20 boron atoms, on polyhedral carboranes such as compounds of the formulas closo--C.sub.2 B.sub.n-2 H.sub.n, closo--CB.sub.n-1 H.sub.n.sup.--, or nido--C.sub.2 B.sub.n-3 H.sub.n.sup.--, on oligomeric peptides constructed from boron-rich α-amino acids, or on boron enriched oligophosphates.